ROUTES OF INFECTION IN TUBERCULOSIS.*

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There is no subject about which there has been so much controversy as about the exact way in which the virus of tuberculosis gains access to the human body and in which it is disseminated after it once has gained entrance. Still when we carefully sift the evidence, we cannot fail to perceive that there is a considerable amount of accurate information at hand, the issues appearing hazy and befogged only through the constant, insistent reiteration of certain convictions by those who in some way or another have become engaged in defending some more or less exclusive theory.

Baumgarten of Tübingen, as you all know, has held for many years that tuberculosis of all forms is most commonly acquired in utero. A transmission of the disease in this case might arise either by means of infected sperm or through the placenta, a primary infection of the ovum before fertilization being very unlikely.

It is a well established fact that the semen of tuberculous individuals contains tubercle bacilli not so very rarely even in the absence of any tuberculous disease of the genital organs, and the possibility certainly exists that by means of such infected semen the developing ovum might become contaminated. This interesting question has been very carefully studied experimentally in rabbits by Friedmann,[1] who comes to the following conclusions: "It should be regarded as proved that tubercle bacilli can enter the fertilized egg cell, that the latter does not perish as a result of the invasion, but may develop into a well-formed animal. In addition, the bacilli transmitted in this way may still be present in certain organs of the new born. On the other hand, it is proved that the rabbit embryo does not become tuberculous in spite of the bacilli it has received in statu nascendi nor the young animal either in the first time of its post-embryonic life." As a result of this investigation the much less rigidly controlled, similar positive experiments of Karlinski[2] on goats fall to the ground, especially since in his animals the infection appears to be closely associated with the alimentary tract. The possibility of a direct infection of the fetus from a tuberculous father may therefore be safely excluded from practical consideration.

As a result of Schmorl's[3] work we know that tuberculous infection of the placenta is much more common than was believed formerly. In 20 placentae of tuberculous mothers he found manifest tuberculosis in three, and few scattered microscopical foci in six. In spite of this fact, evidence of congenital infection in man is extremely rare, and from what we know of the lack of resistance of the tissues of young children to tuberculous infection, we are not at all warranted in assuming that such infections would remain latent for many years to become active at the time of puberty or even later, as has been suggested by some. If infants do become infected, as they naturally sometimes will, their disease makes rapid progress, and at autopsy is found very extensive and widely disseminated, the appearance of the disease being very similar to that which we find in the least resistant animals, like guinea pigs.

There is no question that small children frequently infect themselves with the dirt which gains access to their mouth from their constantly soiled fingers, as emphasized by Volland.[4] Tuberculosis of the tonsils, or cervical glands, or both, is a very common result, and naturally bacilli taken up in this way may be carried either into the lungs or into the alimentary tract, and may set up foci of infection in those regions. However, even in children, it is becoming apparent more and more that the most common mode of infection is by means of the aspiration of infectious material into the lower respiratory tract. Unquestionably a certain percentage of children, variously estimated from about 15% to 25%, become infected by the alimentary route and in these cases the ingestion of bovine tubercle bacilli in contaminated milk seems to play a certain role, but of these same children some acquire a simultaneous or subsequent infection by way of the tonsils or the respiratory canal or both. Such a case of apparently triple infection of a baby nine months old has been described by me in a previous paper.[5] In adults the clear cases of primary intestinal infection are much less common. In a series of cases of my own, for instance, I found them to represent about 6% of the active cases of tuberculosis among adults. The extensions of the process in these cases, if there were any, were largely in the abdominal cavity involving the mesenteric glands, the peritoneum, the genito-urinary tract in the male, the internal genital tract in the female, the adrenals, and the liver. In a comparatively large proportion the fatal termination was due to acute or subacute disseminated tuberculosis.

The original anatomical conception of a primary infection of the lungs in ordinary cases of chronic pulmonary tuberculosis of the adult, has been greatly strengthened not only by more careful anatomical investigation of incipient stages, but also by experimental evidence. Birch-Hirschfeld's[6] anatomical studies which have been amply confirmed by later investigators will always remain classic in this regard. He demonstrated the fact, that in the vast majority of cases fairly large bronchi in the posterior part of the apical portions of the lung are primarily affected. Occasionally only did he find the primary focus in the pulmonary tissue itself, when it is naturally much more difficult to tell in which way exactly the infectious material had en-

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tered this organ. It has also been shown by Schmorl [7] that, when the lungs do become infected by way of the blood current from some other focus, that the distribution and later development of the lesions is entirely different from that in ordinary pulmonary tuberculosis.

The strongest *experimental* evidence which we possess in favor of the frequency of pulmonary infection, is the fact that susceptible animals can be inoculated much more readily by the respiratory than by the intestinal route. The first tentative comparative experiments in this regard were conducted by Gebhardt (1890) and Preyss (1891), but the most careful work in this direction was done by Findel [8] in Flügge's laboratory in 1907. Findel found that, in case of inhalation, 62 tubercle bacilli approximately constituted a certainly infectious dosis for grown guinea pigs, whereas he obtained no results, when 6000 times this number was introduced into the alimentary canal with the food. It seems, that it takes probably a dose one million times larger than the one that kills by the respiratory route, to effect an infection by way of the alimentary canal in these animals, and the same holds true for other species of mammals that have been investigated. In fact, some resistant animals like rats can be infected by the aerial route comparatively easily, by the intestinal practically not at all.

It would also appear from Findel's records, that the disease produced in his guinea pigs by the aspiration of few bacilli resembles pulmonary tuberculosis in man in so far as it is more chronic; the tubercles become larger and show cavitation. The peculiar localization of the lesions at the apices is not reproduced, however.

It is true, that more or less isolated pulmonary lesions have been produced in resistant animals by subcutaneous injection or in susceptible animals by introduction of tubercle bacilli into especially resistant organs like the bladder, but I cannot believe that these experiments are directly applicable to human pathology so far as pulmonary tuberculosis is concerned.

The usual localization of the virus in man in the apices is still a very puzzling problem, in spite of much ingenuity displayed in attempts at finding its solution. Are the tubercle bacilli carried more easily to this part of the lung? If not, is there any special difficulty in eliminating them after they have arrived there? Is perhaps the apical tissue less resistant to tuberculosis than the rest of the pulmonary tissue, on account of a relative anemia, for instance? These are some of the many questions which have been asked. It has been stated in a general way, that the apices in man are poorly ventilated; I believe, without much justification. One is apt to forget, that the lungs are of a wonderfully equally distributed elasticity throughout, which elasticity causes them to expand equally throughout, wherever the pull may be. This extension of the elastic pull all over the lung is greatly facilitated by the fact that they are not fastened on the surface, but slide easily against the chest wall, which movement may be observed very clearly on direct inspection through the intact pleura. As a matter of fact, the apical parts of the lungs of *animals* would appear to be in a much worse condition so far as ventilation is concerned than the same region in man. It is true, they do not project as much above the upper aperture of the thorax, but the breathing in animals is entirely abdominal, whereas in man even in quiet breathing there is some excursion to the upper part of the chest, and yet animals do not develop apical tuberculosis either spontaneously or experimentally.

I am convinced, that ordinarily the apical parts of the lungs in man are as well ventilated if not better than others, the posterior lower parts, for instance; for one reason on account of the shorter reach to the main bronchi. I also cannot believe that even in the erect posture there could be enough of a difference in the circulation in the upper and lower parts of the lung to account for any difference in resistance. That there should be a mysterious lack of resistance in the apical portions to infection in general or to tuberculosis in particular, is equally unproved. The only tangible difference, which remains is this, that in forced expiration air may be driven up into the apices on account of their not being enclosed in the rigid wall of the thorax. In this way infectious material in the bronchi might be thrown up toward the apical parts of the lung. This would mean, that infected material breathed in *in man* would not be carried at once to the finer ramifications of the bronchial tree, but would be deposited in the large bronchi near the bifurcation where it would be received in the bronchial mucus, and in most cases expectorated.* During forced expiration such infected mucus might be carried up by a sudden rush of air into the bronchi of the apices, where an infection may result in the more delicate mucous membrane. This assumption also would give at least some explanation why the infection of the apices so frequently takes place on both sides more or less simultaneously.

One would imagine that the distribution of dust, more especially of coal dust, in the lungs of man, would give some clue in regard to the probable distribution of aspirated infectious material, and it has been claimed, that such dust did accumulate in larger quantity in the apical parts of the lungs. One should not forget, however, that disease often contributes to effect this localization. It is well known that dust of this kind accumulates in areas of chronic inflammation and since the majority of individuals have old scars in their apices, the apices naturally appear more deeply pigmented. The normal distribution of dust can be studied only in perfectly healthy lungs, and we all know, that they are the exception rather than the rule. I have lately looked over a series of such lungs, and I find that under these circumstances the distribution of the coal dust is remarkably general, but that the posterior parts of the lungs are somewhat more deeply stained than the anterior ones, both in upper and lower lobes. There is no striking excess of pigment in the usually infected region in the apices

* That such aspirated material may remain in the bronchi for weeks before being eliminated, is brought out very clearly in Arnold's experiments on dust inhalation. (Arnold: Staubinhalation und Staubmetastase. Leipzig, 1885.)

either in early or late stages of uncomplicated anthracosis.

The attempts by Freund, Hart,[9] and others to deny the general predisposition of the apices to tuberculous infection, and to substitute instead an individual disposition due to disturbances in development or use of the upper aperture of the chest are seriously weakened by the fact that so many individuals, variously estimated from 60% to 90% or more, do contract a tuberculous infection of their apices from which the vast majority recover spontaneously. If the local predisposition by anomalies of the upper aperture of the chest is so widely disseminated in the human race, it loses much of its interest. If it is not a question of favoring infection, but of favoring the development of the disease, then we have other and much more substantial general factors to fall back on than such at best illy defined local conditions, quite apart from the fact that we are fairly certain, that stopping the respiratory excursions rather favors healing of tuberculous processes than otherwise. The well-known experiments of Bacmeister[10] are very interesting, but hardly reproduce conditions such as we find them in man. I may merely state, that in practically all his experiments there were other foci in the lungs of the same age as the one at the apex, in order to point out one of the differences, and there are others which it would lead me too far to enter into on this occasion.

These considerations about the sources of infection in the ordinary cases of tuberculosis may not be of overmuch interest to men engaged in the specialties, still I could not refrain from saying this much, because I am firmly convinced, that the specialist should be possessed of the best knowledge in general medicine first, before undertaking special work in any part of the human anatomy. The human body is a whole first and all the time; it is only we who for our own convenience and often not less to our confusion try to break it up into its component parts. It is one of the most promising signs in the development of modern medicine, that this is being realized more clearly from day to day all over the civilized world.

You are all familiar with the fact that the tonsil is a frequent point of entry for tuberculosis, more especially in children. About 5% of tonsils of children selected at random for examination show histological evidences of tuberculosis and very frequently tubercle bacilli seem to pass through them to the regional lymph glands without producing any local infection. Secondary infections of the tonsils in cases of pulmonary tuberculosis are even more common. Other parts of the upper respiratory and alimentary tract are much less frequently exposed to primary infection.

There are few cases in literature of well authenticated infection with tuberculosis by way of decayed teeth, but the observations of Partsch[11] and Euler[12] prove conclusively that such infections may occur occasionally.

In connection with the usual anatomical seat of the pulmonary infection in a small bronchus, it is interesting to recall, that primary infections of the larynx or trachea and larger bronchi occur once in a while, as one would presume if he accepts the theory of the conveyance of the disease by means of the air. This is well known in case of the larynx, but Gidionsen,[13] Hedinger,[14] Hansemann, and Schmorl have described cases in which the primary focus, usually in the form of a tuberculous ulcer, was in the trachea or one of the main bronchi. In these cases the most careful search for possible primary lesions elsewhere failed to reveal any.

Steward[15] has collected the records of 100 patients with tuberculous infection of the nose. Of these, more than one-half (58%) appeared to be primary clinically. Even if we make allowance for the fact that clinically one cannot exclude a focus elsewhere with certainty, the figure is suggestive. The most common site is the anterior part of the septum. The disease appears as an infiltration with or without ulceration or sometimes in the form of tumor-like masses of slow growth which may project into naso-pharynx. It is especially these tumor-like cases which usually seem to be primary. Tuberculosis of the accessory sinuses, except by extension in nasal lupus, appears to be exceedingly rare.

The relations between the conjunctiva, the lachrymal passages, and the nose being such close ones, it is not surprising that Hinsberg[16] in nine cases of tuberculosis of the nose found disease of the lachrymal passages or of the eye and its surroundings in five.

The mode of infection in tuberculosis of the conjunctiva has been much discussed. Casali[17] on the basis of his experiments, believes that the infection is usually an exogenous one, whereas Stock[18] reports a severe tuberculosis of the conjunctiva in a rabbit 4½ months after intravenous injection of tubercle bacilli. There are several cases on record of what was clinically regarded as primary tuberculosis of the conjunctiva. The most convincing observation of this kind appears to be the one described by Thompson.[19]

For practical purposes it is well to remember that a considerable proportion of cases of empyema of the lachrymal sac are tuberculous (Bribak, 2 in 16; Stock, 6 in 106).

The skin is so well protected, that a primary infection of the same is rather exceptional. We all know of the tubercles in the skin of the hands of pathologists and similar lesions observed in butchers who handle much tuberculous material; but otherwise infection by contiguity from mucous membranes or from tuberculous glands (Jones[20]) or by way of the blood current seems to play a more important role than is commonly believed, except of course in the direct sputum infections of the skin of the hands and elsewhere, which are fairly common in consumptives.

One of the most interesting chapters of my subject is that on the tuberculosis of the middle and inner ear. We can roughly distinguish between tuberculosis of the middle ear in children, tuberculosis of the middle ear in consumptives, and more or less isolated tuberculosis of the mastoid both in children and adults. We owe much of our

knowledge of the pathology of these conditions to Habermann,[21] who did his work in Chiari's laboratory in Prague. He became convinced that the infection in many cases was primarily one of the mucous membrane of the middle ear, and he first gave a clear account of the mode of infection, which undoubtedly is the usual one in consumptives. He said "that the tubercle bacilli attached to small particles of sputum are thrown in coughing through the Fallopian tube into the middle ear." He did not, however, deny the possibility of a hæmatogenous infection. How frequently such hæmatogenous infections occur and under what circumstances has been much discussed without leading to any general agreement. The cases are usually examined so late that it is impossible to still trace the mode of infection. In the cases of tubercular mastoiditis especially, infection by way of the blood current has suggested itself, but it has also been pointed out, that in them tubercular lesions in the mucous membrane of the middle ear are never lacking. The fact that the mucous membrane is often involved primarily, does not in any way prove that the infection must have taken place from the surface. Körner [22] suggests that tubercle bacilli may be carried from infected tonsils by way of the lymph current along the path of the Fallopian tube. Hæmatogenous infection, on the other hand, does not necessarily mean primary infection of the bone; there is no reason why the blood current should not carry the tubercle bacilli to the mucosa as well. If, however, one could prove that certain forms of tuberculosis of the ear were primarily located in the bone, then no other form of infection except the hæmatogenous could be considered. This is what Barnick [23] attempted to do, but he himself does not claim that his investigations are conclusive in this regard, only suggestive. Henrici [24] is much more positive that all cases of tuberculosis of the mastoid in children are hæmatogenous and primarily located in the bone. Brieger [25] in his monograph published in 1913 sums up the situation as follows: "It is probable that the transportation of the tubercle bacilli to the middle ear usually occurs by way of the Fallopian tube. Tubercle bacilli may also enter the *mucosa* of the middle ear by way of the blood current. Hæmatogenous infection in middle ear tuberculosis does not mean primary localization in the bone. It is not proved, that the marrow is the point of origin of certain forms of middle ear tuberculosis." In this last assertion he is undoubtedly correct, but the opposite is just as true and from more general pathological considerations it would be quite remarkable, if tuberculosis of the bone should not occur in the region of the middle ear, especially in children. I do not think, therefore, that it is wise to embrace any too exclusive view on the subject, and although the infection of the ear in consumptives undoubtedly most commonly takes place in the way described by Habermann, in other forms of tuberculosis of the ear the possibility of hæmatogenous infection either of the mucous membrane or of the bone should always be considered.

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Discussion.

Dr. C. F. Welty: As to tuberculosis of the nose, I agree absolutely with what the doctor has to say; about the tonsils, I am also of the same opinion; but when you come to discuss tuberculosis of the ear, there are so many phases that I will probably forget some of the things I want to say.

In the first place, you must bear in mind that tuberculosis of the ear never appears as such. You always have a discharging ear, and grafted on to your discharging ear, your tuberculosis appears; that is worth thinking of a great deal. If the tuberculosis entered by the blood stream you would have a spontaneous tuberculosis of the ear, which as yet we have been unable to diagnose.

Politzer's teaching about acute purulent otitis in tubercular individuals was this: Never allow that otitis to last for more than six weeks. In other words, operate your otitis without mastoid symptoms. The reason that he recommended operation was because of the infection that might follow, and an ear that is infected by tuberculosis will not get well by itself.

Here is another thing that the doctor calls attention to, in regard to the infection of bone. It is a known fact that tubercular ears in tubercular individuals (I am speaking only of proven cases of tuberculosis) usually succumb to meningitis by way

of the labyrinth. Many of the cases of chronic suppurative otitis media that have tuberculosis die if not operated on. Reasoning in such a way, we must believe that it is inoculated by way of the Eustachian tube rather than from the blood stream.

Dr. J. J. Kingwell: I want to state a case which has been in my hands for the last two weeks—a patient with positive tuberculosis of the intestines and lungs. Two weeks ago she had a coughing spell, followed by a severe pain and fullness in the ear. Within two days she sent for me. The canal had the appearance of an acute otitis—drum bulging, hyperemic. I did a paracentesis, but found no pus or serum. It was treated antiseptically. About a week ago I was called to see her again. The mucous membrane was swollen and pus exuded. A swab taken from the same proved positive tubercle bacilli. There are no symptoms of mastoiditis.

Dr. H. S. Moore: Speaking of tuberculosis of the nose or larynx: In a short paper I read a few months ago, I said I really believed it would be proven that there are no primary infections in the larynx; there may be in the nose. I ran a series of 200 cases in the Tuberculosis Clinic of Stanford having this point in mind, and Dr. Clark, the internist, always found the primary lesion elsewhere.

Dr. H. B. Graham: I want to thank Dr. Ophüls for the remarkably clear paper he has presented, and compliment him on the way he stuck to his subject—so much better than we get in our discussion.

I think we have not yet enough data to make a decision as to whether or not primary tuberculosis of the larynx actually occurs. The teaching in Vienna by Albrecht—who made a special study of tuberculosis throughout the body, its routes of infection—was that primary tuberculosis of the larynx was practically unknown, and that if it did occur, it was practically unknown to him. At one period, when he was working in the Allgemeine Krankenhaus, he found that in all cases of tuberculosis of the larynx, he could find healed active or passive lesions in the lungs or other portions of the body, which seemingly antedated the lesions in the larynx.

I believe in most of the cases that we say clinically are cases of secondary tuberculosis of the larynx, the route of infection is by the lungs and bronchial lymph nodes, or by sputum coughed up into the larynx. I simply repeat what Albrecht gave us in our work there, so my remarks are based entirely upon his findings.

I have been much interested in the subject of tuberculosis of the ear and the route of infection there because two years ago I had occasion to operate a case of tuberculosis of the nasopharynx in whom I had some time before made a diagnosis of tuberculosis of the middle ear. The patient was a very healthy woman of 35 who showed no evidences of ever having had tuberculosis of the lungs. She had a typical picture of tuberculosis of one ear which had healed spontaneously, leaving a latent labyrinthitis. When I saw her she had a picture of an acute middle ear tuberculosis on the other side. I hesitated about operating that side because she had already lost the one ear. In the course of my observations I found a granulating area on the vomer. It was limited entirely to the septum and was ulcerated and not lupoid. The lesion was curetted and sent to the microscopist and was returned with the diagnosis of tuberculosis. The lesion in the nasopharynx healed under cauterization and curettement after two months.

I am pretty positive that in this case the route of infection was from the nasopharynx into the ears. I do not believe that was a hematogenous infection; I do not believe, with Dr. Ophuls and the men he has quoted, that the route of infection is hematogenous but by the Eustachian tube.

I had occasion to look over the literature about a year ago for a paper which is to be published very soon on tuberculosis of the middle ear, and in all the literature I never found a case where there was not a very grave doubt of the hematogenous origin of the tuberculous lesion. The cases in the English literature are simply worthless; those in the German literature are very doubtful. It is so easy to believe that the tubercle bacillus may be coughed up into the Eustachian tube that it seems to be stretching the imagination to take that tubercle bacillus through the body in the blood stream.

A STATISTICAL STUDY OF RABIES IN CALIFORNIA.

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Since 1909, and until recently, rabies has been epidemic in California. Despite the dissemination of knowledge in regard to the control of the disease, rabies among animals in California steadily increased. The height of the epidemic has been reached and passed, and rabies may be considered under control except in Modoc and Lassen counties.[1] This is partly due to the peculiar tendency of an epidemic of rabies to spend itself, the measures instituted for control, and the fact that the disease has become endemic in the more populous communities.

The advent of the disease in coyotes in Modoc and Lassen counties, which was accomplished through infection traveling from Oregon and Nevada, was made the basis of a remarkable campaign against these animals by the California State Board of Health. The financial loss in livestock alone in Modoc and Lassen counties from rabies places this disease in the forefront as the enemy of the cattle and sheep men. Therefore, the prompt eradication of rabies is a necessity, both in city and rural communities, because of the serious element of human danger on the one hand, as shown by the large number of deaths from rabies in human beings in California, and from an economic standpoint on the other, as shown by the experience of Modoc and Lassen counties.

RESULTS OF LABORATORY EXAMINATIONS.

Beginning April 1, 1913, and ending March 31, 1914, 427 examinations of the brains of animals for rabies have been made in this laboratory. Of these specimens, nine were in such a state of decomposition as to make examination impossible. Seventy brains gave negative results and 348 were found positive. Three hundred and thirty-nine of the positive cases were diagnosed by the finding of Negri bodies and the balance by inoculation into rabbits and guinea-pigs. The animals affected were as follows: 317 dogs, 14 cows, 11 cats, 4 horses, 1 goat, 1 coyote.

We have received reports of the biting of 235 human beings and 207 animals by the animals proved positive by our examinations. As to months, the highest number of examinations were made in the cold months, December and January. This does not substantiate the popular belief in increased prevalence in summer. Twenty-five posi-

1 Geiger, J. C.: Is Rabies Under Control in California? Cal. State Jour. Med. February, 1916.